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PERIPARTUM CARDIOMYOPATHY(PPCM)- CASE REPORT

Anaesthesiology

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ABSTRACT

Peripartum Cardiomyopathy (PPCM) or dilated cardiomyopathy is defined as systolic heart failure in the last month of pregnancy or within five months of delivery. Its diagnosis is often delayed because its symptoms closely resembles those within the normal spectrum of pregnancy and postpartum period. We report a case of 25 yrs female presenting in intraoperative period during caesarean section. The diagnosis and subsequent management of this entity in post operative period requires a clinician to be aware of clinical presentation of PPCM. This patient recovered well.

KEYWORDS

Peripartum cardiomyopathy, caesarean delivery,anaesthesia, heart failure

Introduction: Peripartum cardiomyopathy(PPCM) is rare and life threatening cardiomyopathy of unknown etiology that affects women in the last month of pregnancy or in the first five months postpartum.[1] The incidence of PPCM ranges from 1:300 to 1:15,000 live births. The definition of PPCM includes four criteria: 1) development of cardiac failure in the last month of pregnancy or within five months of delivery , 2) absence of an identifiable cause for the cardiac failure, 3) absence of recognizable heart disease before the last month of pregnancy and 4) left ventricular (LV) dysfunction (ejection fraction of < 45% or reduced shortening fraction), Fractional shortening <30% and increased left ventricular end diastolic dimensions (LVID)>2.7 cm/m2.[2,3,4]

This article aims to alert obstreticians and anaesthesiologists who as perioperative physicians need to diagnose PPCM in the perioperative period as early as possible and institute aggressive treatment measures to improve outcome.

Case Report

A 25 yrs old female, primigravida who had an uncomplicated full term pregnancy, was posted for emergency LSCS for oligohydramnios- . General examination revealed height 160 cms, weight 75 kgs, pulse rate --86/min reg., BP 126/88 mm of hg in supine position. There was no oedema, cyanosis or icterus. Her systemic examination was CVS: Normal heart sounds, no murmur. RS: Normal breath sounds , no rhonchi or crepts . CNS: NAD . P/A: 36 wks uterine height. We planned surgery under subarachnoid block. She was premedicated with inj Metoclopramide 10 mg+ Ranitidine 50 mg iv and was preloaded with 500 ml RL. Standard monitors were attached ,subarachnoid block was established in sitting position at L3-4 interspace with inj. Bupivacaine (0.5% heavy) 12 mg to achieve a sensory level of T5. Patient was laid supine with wedge under rt. buttock to avoid A-C compression. Supplemented with oxygen 3 lits/min via nasal prongs . BP was monitored every 3 min. IV RL 500 ml was coloaded within next 10 min. 10 mins later , a male baby weighing 2.4 kgs was delivered , with APGAR score of 8/10 at 1 min and 10/10 at 5 min . inj pitocin 2.5 U iv bolus was given after delivery of baby followed by 10 U in infusion of RL to be infused over next 30 mins. Uterus was extracted outside and was sutured and later repositioned inside the peritoneal cavity. Patient developed bradycardia pulse rate dropped to 40/min , this was treated with inj Atropine 0.3 mg iv bolus, pulse rate started rising and patient complained of headach and nausea . She was given inj. Ondensetron 4 mg iv for that. Later pulse rate accelerated to 226/min and B P shot to 180/110 . ECG showed severe tachycardia(narrow complex) with intermittent broad complex ventricular run. Carotid massage was tried initially without any result. Inj xylocard was given 100 mg bolus for ventricular ectopics. Inj dexmeditomidine 50 mic was given as slow iv bolus over next 10 mins. Pulse rate slowly dropped down to 120/min and BP slowly came down to 140/90 mm of hg. Rest of the surgery remained uneventful except for mild headache. Patient was shifted to postoperative recovery area where she started complaining of breathlessness and vomited once. Spo2 started falling to 92% and examination revealed bilateral crepts and rhonchi. Her BP then was 110/70 she was treated with 100% oxygen. Inj lasix 60 mg given iv bolus and patient was shifted to SICU. In SICU her BP was recorded 80/40 and Spo2 dropped to 88% with O2 supplementation of 8 lit/min. Her RR was 30/min with bilateral coarse crepitations . Differential diagnosis of pulmonary embolism /PPCM/ valvular lesion with LVF?? Was thought off. Her ECG, Xray Chest , 2 D echo were advised . Her ECG revealed sinus tachycardia, X ray chest showed signs of pulmonary oedema while 2 D echo revealed EF of 40% with global hypokinesia, FS% 22.64, LVIDed 2.74 cm/m2. She was again given inj lasix 40 mg and was started with ionotropes Noradrenaline along with dobutamine . Her urine output was 1800 ml till then. A central venous line was established through right jugular vein and CVP was measured it came out to be 5 cms of water . NS bolus of 200 ml was given and CVP was rechecked which was 16 cm of water then—it pointed her very poor cardiac status. Fluids were restricted , her vitals were maintained with BP- 90/60, and pulse rate around 90/min and SpO2 around 92%. She was given inj digoxine 0.25 mg slow iv. On post –op day 2, she maintained her BP around 90-100 systolic and her noradrenaline support was withdrawn. On 4 th post –op day 2D echo was repeated her EF improved to 56% , she was haemodynamically stable so was shifted out of SICU. She was continued on tab Dytor 100 mg (spiranolactone)+ tab. Digoxine .25 mg od. Rest of the hospital stay was uneventful and patient was discharged on day 10th.

Discussion:

Peripartum cardiomyopathy : Incidence is less than 0.1% of pregnancies but morbidity and mortality rates are high at 5-32% . The outcome of PPCM is variable . For some women the clinical and electrocardiographic status improves and sometimes returns to normal ; whereas for others , the disease progresses to severe cardiac failure and even cardiac death [3] . Survivors of PPCM often recovers from left ventricular dysfunction ,however they may be at risk of recurrence in subsequent pregnancies . In severe cases ,women deteriorate rapidly with no improvement with medical line of treatment and may require cardiac transplant or die of heart failure ,thromboembolic events and/or cardiac arrhythmias [5]. Overall PPCM occurs in one of every 300-15000 pregnancies.[6,7] However all races can be affected. The etiology of peripartum cardomyopathy is unknown . Recent evidence suggest that this disease is actually a type of

myocarditis arising from an infectious, autoimmune or idiopathic process. A review of literature reveals that endomyocardial biopsies have shown that from 8.8% -78% of women with idiopathic dilated cardiomyopathy in the peripartum period had evidence of myocarditis.[8-11] The disease has been reported more frequently in women with multiple pregnancies, a history of pre-eclampsia and long term oral tocolytics(>4wks) . A fluid overload in pregnancy may cause subclinical heart disease, which becomes overt because of the additional haemodynamic stress on the cardiovascular system. The onset of peripartum cardiomyopathy usually occurs in the first three months postpartum. Approximately 75% are diagnosed within 1st month postpartum and out of that 45% present in first week in USA statistics.[12] Demakis et al.[2] established the criteria for diagnosis of peripartum cardiomyopathy in 1971:the heart failure must become manifest in the last month of pregnancy or within 5 months of delivery, and no other etiology for heart failure can be found.

Women usually present with dyspnea, cough, fatigue, orthopnea, paroxysmal nocturnal dyspnea, palpitations, chest discomfort and sometimes chest or abdominal pain . Physical examination frequently demonstrates increased blood pressure and jugular venous pressure , cardiomegaly, third heart sound , mitral and/or tricuspid valve regurgitation , pulmonary rales , peripheral oedema, hepatomegaly and less commonly embolic events, haemoptysis and ascitis. An electrocardiogram may demonstrate no abnormality or may show nonspecific ST -T changes , evidence of left ventricular hypertrophy , prolongation of PR or QRS intervals, low voltage and occasionally left bundle branch block.[12] Chest film reveals typical signs of congestive heart failure, including cardiomegaly, pulmonary venous congestion, interstitial oedema and occasionally pleural effusion. Echocardiography demonstrates dilated cardiomyopathy involving all four chambers , but primarily the left heart. Little difference in the left ventricular diamensions is noted from diastole to systole. Normal wall thickness with diffuse symmetrical hyokinesia with high intracardiac pressure with low cardiac output are very characteristics of peripartum cardiomyopathy. Left atrial enlargement with mitral regurgitation is common and there is high incidence of mural thrombi.

Treatment of heart failure includes measures to reduce preload, afterload and improve cardiac contractility using diuretics, nitrates and digoxin. Cardiogenic shock requires the use of ionotropes , intensive haemodynamic monitoring and noninvasive or invasive ventilation. ACE inhibitors can be used in the postpartum period and beta blockers once the condition stabilises.

Similar case of cardiac arrest in undiagnosed dilated cardiomyopathy presenting for emergency LSCS has reported [13] where patient developed severe tachycardia followed by ventricular fibrillation and cardiac arrest and was revived with immediate defibrillation . Mohammad Shafiq et al [14] has reported a similar case of emergency LSCS presented with heart failure and hypoxia in immediate post operative period progressed to cardiac arrest but successfully revived later diagnosed as PPCM.

Conclusion

Peripartum cardiomyopathy is an uncommon disease that is often fatal for the patient, but fetal outcome is quite a good . The physician who cares for the patient with peripartum cardiomyopathy should counsel her about future pregnancies , breast feeding ,and contraception. Heart failure in post operative period should be diagnosed and treated promptly

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